Refine Search

Search Results -

Terms	Documents
L1 and trail	3

Database:
US Pre-Grant Publication Full-Text Database
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Derwent World Patents Index
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Search: L3

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Recall Text | Clear | Interrupt

Search History

DATE: Thursday, April 19, 2007 Purge Queries Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT; PLUR=YES; OP=OR			
L3	L1 and trail	3	L3
L2	L1 and (trail and antiprogestin)	0	L2
L1	Kumar-vijay.in.	52	L1

END OF SEARCH HISTORY

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? s androgen(5n)(independen? or insensitiv?)
          100035  ANDROGEN
         1549213  INDEPENDEN?
          117085  INSENSITIV?
      S1   11511  ANDROGEN(5N)(INDEPENDEN? OR INSENSITIV?)
? s trail(5n)resistan?
           19960  TRAIL
         1740950  RESISTAN?
      S2    1448  TRAIL(5N)RESISTAN?
? s s1 and s2
           11511  S1
            1448  S2
      S3       7  S1 AND S2
? rd

>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
      S4       3  RD  (unique items)
? t s4/3,k,ab/1-3

 4/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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15595825   PMID: 15897871
  Methylseleninic acid sensitizes prostate cancer cells to TRAIL-mediated
apoptosis.
  Yamaguchi Kenya; Uzzo Robert G; Pimkina Julia; Makhov Peter; Golovine
Konstantin; Crispen Paul; Kolenko Vladimir M
  Department of Urological Oncology, Fox Chase Cancer Center, Philadelphia,
PA 19111, USA.
  Oncogene (England)  Sep 1 2005,  24 (38)  p5868-77,  ISSN 0950-9232--
Print  Journal Code: 8711562
  Publishing Model Print
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  Tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) is a
cytotoxic agent that preferentially induces apoptosis in a variety of human
cancer cells. Unfortunately, some tumor cells remain ***resistant*** to
  ***TRAIL***  . Therefore, agents that sensitize malignant cells to
TRAIL-mediated cell death might be of particular importance for the
development of novel antitumor therapeutic regimens. Recent studies
establish a critical role of selenium in prostate cancer prevention in
vitro and in vivo. Here, we demonstrate that concomitant administration of
TRAIL and methylseleninic acid (MSA) produces synergistic effects on the
induction of apoptosis in androgen-dependent LNCaP and androgen
- ***independent***  DU-145 prostate cancer cells. MSA rapidly and
specifically downregulates expression of the cellular FLICE inhibitory
protein, a negative regulator of death receptor signaling. In addition, we
demonstrate that the synergistic effects of MSA and TRAIL result from the
activation of the mitochondrial pathway-mediated amplification loop.
Addition of MSA effectively blocked TRAIL-mediated BAD phosphorylation at
Ser112 and Ser136 in DU-145 cells and was accompanied by induction of the
mitochondrial permeability transition and release of apoptogenic cytochrome
c and Smac/DIABLO proteins from the mitochondria and into the cytosol.
These results suggest that selenium-based dietary compounds may help to
overcome resistance to TRAIL -mediated apoptosis in prostate
cancer cells.

  ... preferentially induces apoptosis in a variety of human cancer cells.
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Unfortunately, some tumor cells remain ***resistant*** to ***TRAIL*** . Therefore, agents that sensitize malignant cells to TRAIL-mediated cell death might be of particular...

... of TRAIL and methylseleninic acid (MSA) produces synergistic effects on the induction of apoptosis in androgen-dependent LNCaP and ***androgen*** - ***independent*** DU-145 prostate cancer cells. MSA rapidly and specifically downregulates expression of the cellular FLICE...

... into the cytosol. These results suggest that selenium-based dietary compounds may help to overcome resistance to TRAIL-mediated apoptosis in prostate cancer cells.

4/3,K,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

13490190 PMID: 11759069

Actinomycin D and gemcitabine synergistically sensitize androgen-***independent*** prostate cancer cells to Apo2L/TRAIL-mediated apoptosis.

Zisman A; Ng C P; Pantuck A J; Bonavida B; Belldegrun A S
Department of Urology, UCLA School of Medicine, University of California at Los-Angeles, 90095, USA.

Journal of immunotherapy (Hagerstown, Md. - 1997) (United States) Nov-Dec 2001, 24 (6) p459-71, ISSN 1524-9557--Print Journal Code: 9706083
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The cytotoxic efficacy and kinetics involved in sensitization of Apo2L/TRAIL-resistant, androgen-independent prostate cancer cells to Apo2L/TRAIL or tumor necrosis factor-alpha or Fas ligand-mediated apoptosis were tested using subclinical concentrations of actinomycin D, paclitaxel, cisplatinum, gemcitabine, and radiation in CL-1, LNCaP, DU-145, and PC3 prostate cancer cell lines. CL-1 cells expressed all four Apo2L/TRAIL receptors and were resistant to Apo2L/TRAIL -mediated apoptosis (1-5,000 ng/mL) and to the sensitizers when given alone. Pretreatment with actinomycin D followed by Apo2L/TRAIL or tumor necrosis factor-alpha or anti-Fas CH-11 monoclonal antibody, but not in the reverse order, induced apoptosis in all cell lines. Synergistic sensitization in CL-1 cells was shown also with gemcitabine but not with cisplatinum, VP-16, paclitaxel, or radiation. Incubating the Apo2L/TRAIL-resistant CL-1, LNCaP, DU-145, and PC3 cell lines with 100 ng/mL actinomycin D for 4 hours followed by Apo2L/TRAIL for 24 hours resulted in 45.4 +/- 10.3%, 58.8 +/- 3.6%, 53.4 +/- 1.4%, and 84.2 +/- 8.4% apoptosis, respectively. Prolonging the sensitization time to 24 hours followed by 20 hours of incubation with Apo2L/TRAIL further enhanced the killing activity against CL-1 cells to 89 +/- 1% (delta = 60%, synergistic ratio = 3.1). This killing has a biphasic pattern that was contributed to by apoptosis (83%) and necrosis (17%) at 10 hours (peak) and 40% and 60%, respectively, at 20 hours. These results suggest that prostate cancer cells' resistance to Apo2L/TRAIL -mediated apoptosis can be reversed and synergy is achieved by sensitization of tumor cells with subclinical concentrations of actinomycin D or gemcitabine and may be useful clinically for the treatment of metastatic hormone- and drug-refractory prostate cancer.

Actinomycin D and gemcitabine synergistically sensitize androgen-***independent*** prostate cancer cells to Apo2L/TRAIL-mediated apoptosis.
The cytotoxic efficacy and kinetics involved in sensitization of Apo2L/TRAIL-resistant, androgen-independent prostate

cancer cells to Apo2L/TRAIL or tumor necrosis factor-alpha or Fas ligand-mediated...

... DU-145, and PC3 prostate cancer cell lines. CL-1 cells expressed all four Apo2L/TRAIL receptors and were resistant to Apo2L/ TRAIL -mediated apoptosis (1-5,000 ng/mL) and to the sensitizers when given alone. Pretreatment...

... also with gemcitabine but not with cisplatinum, VP-16, paclitaxel, or radiation. Incubating the Apo2L/ ***TRAIL*** - ***resistant*** CL-1, LNCaP, DU-145, and PC3 cell lines with 100 ng/mL actinomycin D...

... and 40% and 60%, respectively, at 20 hours. These results suggest that prostate cancer cells' resistance to Apo2L/TRAIL -mediated apoptosis can be reversed and synergy is achieved by sensitization of tumor cells with...

4/3,K,AB/3 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

12895715 Genuine Article#: 831NY Number of References: 41
Title: X-linked inhibitor of apoptosis protein inhibition induces apoptosis and enhances chemotherapy sensitivity in human prostate cancer cells (ABSTRACT AVAILABLE)
Author(s): Amantana A; London CA; Iversen PL; Devi GR (REPRINT)
Corporate Source: AVI Biopharma Inc,Canc & Endocrine Program,4575 SW Res Way,Suite 200/Corvallis//OR/97333 (REPRINT); AVI Biopharma Inc,Canc & Endocrine Program,Corvallis//OR/97333(grdevi@avibio.com)
Journal: MOLECULAR CANCER THERAPEUTICS, 2004, V3, N6 (JUN), P699-707
ISSN: 1535-7163 Publication date: 20040600
Publisher: AMER ASSOC CANCER RESEARCH, 615 CHESTNUT ST, 17TH FLOOR, PHILADELPHIA, PA 19106-4404 USA
Language: English Document Type: ARTICLE
Abstract: apoptosis and enhanced chemotherapy sensitization in ***androgen*** -refractory prostate cancer cells. ***Androgen*** - insensitive prostate cancer cells are highly resistant to several chemotherapeutic drugs and are characterized by the appearance of apoptosis-resistant cells. In this study, we identified the critical role of X-linked inhibitor of apoptosis protein (XIAP), a potent antiapoptotic factor, in conferring chemotherapy resistance in an ***androgen*** - ***insensitive*** DU145 human prostate cancer cell line. Results reveal that DU145 cells were highly resistant to cisplatin, but this resistance was overridden when the cells were treated for a prolonged time (>96 hours) with cisplatin (IC50 = 27.5 to 35.5 mumol/L). A decrease in levels of XIAP and Akt/phospho-Akt and an increase in caspase-3 activity were identified to be key factors in cisplatin sensitivity (40% to 55% decrease in cell viability) at later time points. In contrast, tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) treatment caused a 40% to 50% decrease in cell viability within 6 hours (IC50 = 135 to 145 ng/mL). However, increasing concentrations or prolonged treatment with TRAIL did not change drug potency. A significant increase in caspase-3 activity was observed with TRAIL treatment with no apparent change in XIAP levels. Specific inhibition of XIAP expression using an antisense XIAP phosphorodiamidate morpholino oligomer induced apoptosis and increased caspase-3 activity. Combination of cisplatin with XIAP antisense potentiated cisplatin sensitivity by decreasing the IC50 from > 200 mumol/L with cisplatin alone to 9 to 20 mumol/L and decreasing incubation time required for activity from 96 to 24 hours. Similarly, TRAIL in combination with XIAP antisense phosphorodiamidate morpholino oligomer enhanced TRAIL potency by 12- to 13-fold. In conclusion, abrogation of XIAP expression is essential for therapeutic apoptosis

and enhanced chemotherapy sensitization in androgen-refractory prostate cancer cells.

Abstract: apoptosis and enhanced chemotherapy sensitization in ***androgen*** -refractory prostate cancer cells. ***Androgen*** -insensitive prostate cancer cells are highly resistant to several chemotherapeutic drugs and are characterized by the...

...inhibitor of apoptosis protein (XIAP), a potent antiapoptotic factor, in conferring chemotherapy resistance in an androgen- ***insensitive*** DU145 human prostate cancer cell line. Results reveal that DU145 cells were highly resistant to...

...Identifiers--LIGAND-MEDIATED APOPTOSIS; C-MYC; UP-REGULATION; XIAP; RESISTANCE; EXPRESSION; TRAIL; LNCAP; GENE; IAP

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